“Hangover Prevention" Gene Important for Mitigation of Acrylics Also Important for Enhancing and Preserving Facility for Creativity and Mitigating Acrylic Buildup from Other Sources Such as Chemical Fumes; Implications for Common Causal Pathway for both Alcohol-Induced IQ-Reduction and the Creativity Enhancement Associated with Historical Alcohol Abuse

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Simon Edwards
Research Acceleration Initiative

Introduction

Neurological studies from within the past ten years have determined that brain damage associated with alcohol abuse is driven only in part by the induction of cell apoptosis in the brain i.e. "Killing brain cells," and is driven moreso by causing changes to the preferred pathway that signals in the brain take to arrive at their destination. These pathways, known as axons, are plastic and able to change in both the short and long-term.

Abstract

The mechanism through which alcohol abuse gradually shifts axonal pathways from routes that are more direct to routes that are less direct, effecting a defacto reduction in IQ, is currently unknown. It has, however, been observed that the re-introduction of alcohol results in the temporary resumption of the use of the historical pathway and a restoration of IQ. Not only may this be a factor in the habit-forming effect of alcohol, it may provide a clue as to what is going on with regard to IQ reduction.

Acrylics, which can harmfully accumulate in the body as the result of ethanol consumption, are chemical cousins to ethanol. These compounds tend to accumulate as a result of cell apoptosis events throughout the body; events made more common when alcohol is present in the bloodstream. Acrylics, like ethanol, can cross the blood-brain barrier and effect axonal shifting.

To venture a hypothesis at what is occurring with regard to the alcohol-associated axonal shifting and reversion phenomenon, inspiration comes from an unlikely source in the form of noticing the absence in East Asian genotypes of genes that facilitate the production of "hangover prevention" enzymes. It has been long-observed and recently scientifically confirmed that individuals of East Asian stock will suffer from hangovers of greater intensity than Europeans from relatively modest drinking and a deficit of enzymes that break down acrylics have been implicated in these hangovers. Incidentally, oral-route treatments to prevent alcohol abuse have been available for several years now based upon inhibiting these genes so as to induce hangovers purposefully in the event that alcohol is consumed.

Acrylics, notably, can also enter the body as the result of inhalation of chemical fumes and these fumes have been known to cause headaches, particularly in individuals of East Asian descent. East Asians have been known to pay extra money to have "new car smell" removed from their automobiles when purchasing a new vehicle; an odor that most people of European descent

find unoffensive and even pleasant. This odor is the result of chemical leeching of the compound used to treat the dashboards of new cars; an acrylic compound.

Since there are no nerve endings in brain tissue, how, then, are headaches induced by the simple inhalation of a chemical? Many medical professionals will claim to have an answer to this question but they cannot in truth claim to know because no one has bothered to research that particular problem. Understanding that problem is is critical to understanding how electrical signals may be blocked or redirected by acrylics and even connects to the topic of migraine headache dynamics as I laid them out in my publication of a couple of years ago on the topic.

In order for chemical fumes to cause headaches, the chemical must enter the bloodstream and subsequently enter the spinal cord tissue. The acrylics that cause these headaches must accumulate (either due to the concentration of the fumes being dangerously high or an inability to enzymatically break down the compounds) within those cord tissues and focus and reflect electrical signals so that energy from several fibers may be redirected down a single fiber, resulting in an excess of charge that is interpreted as pain. As with migraine pain, the base cause of this pain this pain is the transmission of excessive amperage up particular fibers of the spinal cord. In migraines, this excess of electricity is sparked by ectopic deposition of brain tissue during fetal development in small clusters that are difficult to differentiate from spinal tissue within the cord. With headaches caused by acrylic fumes, acrylic molecules act like a lens that reduces electrical transmission down intended pathways and increases it in undesired pathways. Thus, the signal associated with six pathways might be shunted momentarily onto one, leading to an overload interpreted by the brain as a headache.

In any hangover, acrylics linger in the neural tissues for longer than the ethanol itself. Thus, even with a natural ability to process acrylics, a European experiencing a hangover will spend most of the next day with measurable levels of acrylics in their bodies, even though they are technically sober. These acrylics inhibit mental performance generally and induce a general dysphoria, both of these caused by the dithering of electrical signals down multiple pathways and pathway dithering being associated with being in distress.

The ability of an acrylic molecule to permute a neural pathway in the long term relies upon its presence in the brain for the bulk of any given 24 hour period, and not so much because of ethanol's presence for, say, a 12-hour period. To be clear, it is not ethanol that causes the axonal shift directly; it is acrylic. Over a period of years, these acrylic molecules do to the brain chemically what mental exercises, sc. "plasticity exercises" might accomplish with the key difference being that instead of restructuring axons in way that enhances specific abilities, acrylics de-train established pathways and generate new pathways that serve no particular purpose. This can be characterized as an IQ-reduction since the metrics we use to measure IQ reward high performance at specific tasks. The count of neurons, however, remains largely unchanged. The change associated with adult-onset alcohol-associated performance deficits is analogous to changing an Application-Specific Integrated Circuit into a more traditional CPU. Although a CPU may

not be as well-suited for specific processing tasks as an ASIC, the variety of tasks it may achieve is greater in diversity and its performance is more standardized between broad categories. It should be noted, however, that the type of damage associated with childhood alcohol exposure and FAS may not be so easily overcome due to systemic damage. These facts should also not be interpreted as a suggestion that any given individual's alcoholism might result in being converted into the next Ernest Hemingway nor is it an endorsement of alcohol consumption.

Ethanol, being a chemical cousin to acrylics, can enhance conduction of electrical signals and ease the flow of energy down pathways rendered disused by chronic acrylic saturation. Instead of conducting and redirecting energy like an acrylic, ethanol may be enhancing conduction without redirection due to structural differences between the chemicals. The conductive capacity of both of these molecules may be attributed to ionization of their outboard hydrogen atoms and the unique tendency of acrylic compounds to redirect energy is attributable to its curvature and its ability to link together with other acrylic molecules in a chain structure (whereas ethanol will not "chain" with itself) that still tends to be positively ionized on the exterior and thus conductive.

Where does creativity come into play in all of this? How does the presence or absence of acrylics in varying quantities affect neural development in the short and long term? How is all of this connected?

The presence of small quantities of acrylic compounds (not associated with alcohol consumption) associated with ordinary cell apoptosis but a lack of ability to efficiently break down these compounds (as in East Asians) over the course of a lifetime may result in axon formations that have a lesser tendency than the European genotype toward deviation due to any cause. The acrylics, in their case, block the establishment of irregular pathways and result in more homogeneous pathways. The fact that this is occurring during early development, happens steadily over time, never abates, and involves quite modest levels of acrylic means that for an East Asian non-drinker, acrylics help to forge a very different neurological structure than does alcohol abuse-associated acrylic exposure which tends to be acute, severe, and detrimental whereas that associated with "being Asian" is tempered by time.

Creativity, I submit, is rooted in having access to signals from the "highest" regions of the brain (those bordering the meninges) where axons tend to be more narrow but larger in number, voltages are higher but amperage is lesser and where tissue convolution increases surface area, but even more importantly, generates substantial fields in which electrons tend to pass through the magnetic wakes of other electrons moving in opposing directions (skyrmion fields;) boosting processing capacity by adding access to information that will become available (in the future) and incorporating those computational results in the present in order to speed the whole process along.

Conclusion

If the above deductions are accurate, we can therefore conclude that acrylics

may be the force majeure in determining the brilliance of Hemingway as well as his dysfunction, the learning difficulties of those who suffer from childhood alcohol exposure's long-term effects, the tendency toward intellectual differences between people of different racial backgrounds and most likely much more.